

### AMENDMENTS TO THE SPECIFICATION

Amend the paragraph at page 12, lines 15 to page 13, line 6 as follows:

One example of an algorithm that is suitable for determining percent sequence identity and sequence similarity is the BLAST algorithm, which is described in Altschul *et al.* (1990) *J. Mol. Biol.* 215: 403-410. Software for performing BLAST analyses is publicly available through the National Center for Biotechnology Information (~~http://www.ncbi.nlm.nih.gov/~~) ([www.ncbi.nlm.nih.gov](http://www.ncbi.nlm.nih.gov)). This algorithm involves first identifying high scoring sequence pairs (HSPs) by identifying short words of length W in the query sequence, which either match or satisfy some positive-valued threshold score T when aligned with a word of the same length in a database sequence. T is referred to as the neighborhood word score threshold (Altschul *et al.*, *supra*). These initial neighborhood word hits act as seeds for initiating searches to find longer HSPs containing them. The word hits are then extended in both directions along each sequence for as far as the cumulative alignment score can be increased. Cumulative scores are calculated using, for nucleotide sequences, the parameters M (reward score for a pair of matching residues; always > 0) and N (penalty score for mismatching residues; always < 0). For amino acid sequences, a scoring matrix is used to calculate the cumulative score. Extension of the word hits in each direction are halted when: the cumulative alignment score falls off by the quantity X from its maximum achieved value; the cumulative score goes to zero or below, due to the accumulation of one or more negative-scoring residue alignments; or the end of either sequence is reached. The BLAST algorithm parameters W, T, and X determine the sensitivity and speed of the alignment. The BLASTN program (for nucleotide sequences) uses as defaults a wordlength (W) of 11, an expectation (E) of 10, M=5, N=-4, and a comparison of both strands. For amino acid sequences, the BLASTP program uses as defaults a wordlength (W) of 3, an expectation (E) of 10, and the BLOSUM62 scoring matrix (*see* Henikoff & Henikoff (1989) *Proc. Natl. Acad. Sci. USA* 89:10915).

Amend the paragraph at page 72, lines 6-15 as follows:

The mouse cDNA, SEQ ID NO:1, is used to isolate a cosmid containing chicken hg.. The hg homolog was checked by DNA sequencing and the gene mapped by fluorescent in-situ hybridization ("FISH") onto chicken metaphase spreads to chicken chromosome 1 (Smith *et al.* *Mamm Genome* 11:706-709, 2000). Markers were developed based on the chicken genomic sequence and

mapped onto a reference genetic linkage map of chicken (the map being available at the Web site <http://www.ri.bbsrc.ac.uk/>) [www.ri.bbsrc.ac.uk](http://www.ri.bbsrc.ac.uk/)). Interestingly *Raidd/Cradd* maps to the approximate location of a growth QTL in broiler chickens (Groenen *et al. Anim. Biotechnology* 8:41-46, 1997). The hg marker can be used to genotype F2 progeny of the cross to confirm linkage to the growth QTL.

Amend the paragraph at page 73, lines 17 through page 74, line 4 as follows:

The study of mammalian growth-control genes is essential for elucidating the mechanism of growth at the tissue, organ or whole-body level. The high growth (hg) mutation is a unique overgrowth model in that it causes a 30-50% increase in postweaning growth without resulting in obesity (Bradford and Famula (1984) *Genet. Res.* 44: 293-308). High growth (HG) mice have increased plasma IGF1 (Corva and Medrano (2000) *Physiol. Genomics* 3: 17-23; Medrano *et al.* (1991) *Genet. Res.* 58: 67-74) and decreased plasma and pituitary GH (Medrano *et al.* (1991) *Genet. Res.* 58: 67-74) suggesting that the causal mutation influences growth through deregulating the GH/IGF1 system. We have shown (Horvat and Medrano (1995) *Genetics* 139: 1737-1748) that hg is not an allele of Gh or Igfl and that it is located within a 500-kb deletion in mouse chromosome 10 (Horvat and Medrano (1998) *Genomics* 54: 159-164). Here we used comparative mapping to identify positional candidates for hg. The mouse hg region was previously mapped (Horvat and Medrano (1998) *Genomics* 54: 159-164) to a genetic interval of 100 to 103 cM from the top of human chromosome 12. Human expressed sequence (EST) clones from this region were selected from the human gene map (<http://www.ncbi.nlm.nih.gov/>) ([www.ncbi.nlm.nih.gov](http://www.ncbi.nlm.nih.gov/)) and used as probes on blots containing mouse BAC clones from the hg region. Southern analysis was performed using standard procedures (Sambrook *et al.* (1989) *Molecular cloning: a laboratory manual. 2nd ed.*, Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY.) under the lower stringency hybridization temperature (55 °C).